218 Temperature

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Definition

Normal body temperature is considered to be 37°C (98.6°F); however, a wide variation is seen. Among normal individuals, mean daily temperature can differ by 0.5°C (0.9°F), and daily variations can be as much as 0.25 to 0.5°C. The nadir in body temperature usually occurs at about 4 A.M. and the peak at about 6 P.M. This circadian rhythm is quite constant for an individual and is not disturbed by periods of fever or hypothermia. Prolonged change to daytime-sleep and nighttime-awake cycles will effect an adaptive correction in the circadian temperature rhythm. Normal rectal temperature is typically 0.27° to 0.38°C (0.5° to 0.7°F) greater than oral temperature. Axillary temperature is about 0.55°C (1.0°F) less than the oral temperature.

For practical clinical purposes, a patient is considered febrile or pyrexial if the oral temperature exceeds 37.5°C (99.5°F) or the rectal temperature exceeds 38°C (100.5°F). *Hyperpyrexia* is the term applied to the febrile state when the temperature exceeds 41.1°C (or 106°F). *Hypothermia* is defined by a rectal temperature of 35°C (95°F) or less.

Technique

Measurement of temperature along with other vital signs should be made with each new patient visit and on a fixed schedule during hospitalization. The glass thermometer is probably the instrument used most frequently. For cooperative patients, the oral glass thermometer is recommended because of its convenience and patient acceptance.

The oral temperature is measured with the probe placed under the tongue and the lips closed around the instrument. The patient should not have recently smoked or ingested cold or hot food or drink. Oxygen delivered by nasal cannula does not affect the accuracy of the measurement. Three minutes is the time commonly quoted for accurate temperature measurement, but it is wise to wait at least 5 minutes. If the reading is abnormal, the thermometer should be replaced for 1-minute intervals until the reading stabilizes.

Rectal thermometers are indicated in children and in patients who will not or cannot cooperate fully. Continuous, frequent temperature measurements can be made by rectal probe and thermocouple connected to a recording device or by repeated glass thermometer measurements in axilla or groin folds. Rectal temperature is measured with a lubricated blunt-tipped glass thermometer inserted 4 to 5 cm into the anal canal at an angle 20° from the horizontal with the patient lying prone. Three minutes dwell time is required.

Electric digital thermometers are more convenient than glass instruments because the probe cover is disposable, response time is quicker (allowing accurate measurements within 10 to 20 seconds), and there is a signal when the rate of change in temperature becomes insignificant.

Reset the glass or electric device to below 35°C (95°F) before each measurement. When hypothermia is suspected, a rectal probe and thermocouple capable of measuring as low as 25°C is essential.

In certain circumstances, it might be important to observe the patient continuously for 15 minutes before and during the measurement of temperature. This would help eliminate the possibility of artifactual readings caused by cold or hot substances taken orally, by smoking, or by surreptitious manipulation of the thermometer. Measurements made by electric devices are less easily influenced by manipulation of the instrument.

Palpation of the skin in the diagnosis of fever is highly unreliable. The presence of fever is underestimated by palpation in 40% of individuals, even when the measured temperature is as high as 39°C (102.2°F).

Patients with fever usually exhibit warm, flushed skin, tachycardia, involuntary muscular contractions or rigors, and sweating or night sweats. Piloerection and positioning of the body in an attempt to minimize exposed surface area are also seen. Occasionally these signs are absent or minimal, and dry, cold skin or extremities are detected in spite of a significant rise in core temperature.

Basic Science

A fine balance between heat production and loss is maintained imperceptibly in the normal individual. In health, the hypothalamic thermoregulatory center monitors internal temperature changes from core thermoreceptors and surface changes from skin thermoreceptors. The center responds to any changes in heat production or ambient temperature that would cause minor deviations from the body temperature "set point" of 37°C (98.6°F). Production of body heat is primarily the result of conversion of chemical energy in foods to heat by metabolic and mechanical mechanisms. Cellular oxidative metabolism produces a constant, stable source of heat. Mechanical muscular contraction results in bursts of heat when needed. Heat produced is conserved by vasoconstriction and diversion of blood flow away from the skin.

Dissipation of heat depends on vasomotor changes that regulate blood flow to the skin and mucous membranes and sweating. Heat is lost at the skin surfaces by the mechanisms of convection, radiation, and evaporation. Dissipation by convection is more efficient when ambient wind current is increased; evaporation is the primary mechanism in high ambient temperatures, unless the atmosphere is saturated with water vapor. Some heat is dissipated by breathing (panting). Heat loss either by conduction through the gastrointestinal (GI) tract via ingestion of cold food and drink or by immersion in cold water is not normally an important mechanism.

A decrease in metabolism, an abnormality in mechanical muscular function, or exposure to ambient temperatures below the normal body temperature may result in *hypothermia*. At a temperature of 32°C (89.6°F), oxygen consumption decreases as a function of hypometabolism, the oxygen dissociation curve shifts to the left so that less oxygen is given up to the tissues, and there is a generalized inhibition of enzyme activity.

Excessive exposure to high ambient temperatures, an increase in heat production (either by increased metabolism or, more often, by increased muscular work) or loss of the ability to dissipate sufficient body heat may result in *hyperthermia*. The hypothalamic "set point" is not disturbed in persons suffering from hyperthermia. The problem is one of overwhelming heat production or inadequacy of heat loss mechanisms. Exercise and heavy work may be responsible for production of heat that raises core temperature 1 to 1.5°C (2 to 3°F), but the temperature usually returns to normal within 30 minutes of cessation of exertion. Overinsulation or exposure to ambient temperatures greater than 37.8°C (100°F), especially in conditions of 100% water vapor pressure and dehydration, interfere with the normal mechanisms for heat dissipation.

Fever, or pyrexia, is the result of the thermoregulatory mechanisms' response to an elevated set point. The set point is raised when acted on by endogenous pyrogen, a substance liberated by leukocytes when they interact with exogenous pyrogens such as microorganisms, nonmicrobial antigens, or certain steroid hormones. Endogenous pyrogen is a protein of 15,000 daltons produced by neutrophils, eosinophils, monocytes, Kupffer cells, and alveolar macrophages, when they are exposed to exogenous pyrogens. Endogenous pyrogen is closely related or identical to lymphokines such as interleukin 1, leukocyte activating factor, and leukocyte endogenous mediator. When endogenous pyrogen is liberated into the bloodstream, it interacts with the preoptic regions of the anterior hypothalamus and raises the thermoregulatory set point to a variable degree, but usually not greater than 41.1°C (106°F). If endogenous pyrogen is placed directly into the cerebral ventricles, high fevers can be induced with concentrations 10- to 100-fold less. Hyperpyrexial states (greater than 41.1°C) may be produced by this direct mechanism. Endogenous pyrogen causes increased firing of hypothalamic, thermosensitive neurons, resulting in the augmentation of heat conservation and production mechanisms, with resultant fever. Moderate increases in the set point are satisfied by heat-seeking behavior, peripheral vasoconstriction, and increased metabolic rate. For marked increases in set point, these mechanisms of heat production and conservation are augmented by mechanical conversion of chemical energy to heat by muscular shivering (rigors). Chilliness felt by the patient whose fever is rising is probably caused by a central perception both of a demand to raise central core temperature and of cold receptors in the skin due to peripheral vasoconstriction.

The molecular mechanisms that mediate the interaction of endogenous pyrogen, the hypothalamus, and effector mechanisms resulting in fever are not completely understood. Prostaglandins of the E series are thought to play a role in excitation of thermosensitive neurons of the hypothalamus. Monoamines are present in high concentrations at that thermosensitive site. Cyclic nucleotides have also been implicated as intermediates induced or released by endogenous pyrogen.

Even during febrile states, the normal diurnal fluctuations in temperature are maintained, although sometimes by extreme mechanisms. For instance, marked muscular activity (rigors) may herald the late afternoon or evening temperature spike in the febrile person, while profuse soaking sweats may be required to achieve the early morning nadir of the circadian temperature rhythm.

Clinical Significance

Closely regulated temperature is imperative for normal and efficient functioning of organ systems. Drastic and irreparable changes in organ structure and function can occur when body temperature falls below 32.2°C (90°F) or rises above 41.1°C (106°F). Lesser changes result in confusion, delirium, seizures, or cardiorespiratory embarrassment, depending on the physical status and age of the host. However, one could point to remarkable recovery of patients with temperature documented below 18°C (65°F) or above 44.5°C (112°F).

Hypothermia can be due to infection and bacteremia, ethanol or drug ingestion, exposure, a central nervous system event, cachexia from malignancy or malnutrition, gastrointestinal bleeding, or endocrine deficiencies such as panhypopituitarism, myxedema, Addison's disease, uremia, and hypoglycemia. In some patients with these diseases, the febrile state might not be recognized because they will raise their temperature to less than 37.2°C (99°F). The early stage of hypothermia (35° to 32.8°C; 95° to 91°F) is marked by an attempt to react against chilling including shivering, increased blood pressure and pulse, vasoconstriction, and diuresis. An intermediate stage (32.2° to 24°C; 90° to 75°F) is characterized by decrease in metabolism; drop in pulse, blood pressure, and respiration; muscular rigidity; a fine tremor; and respiratory and metabolic acidosis. At a third stage, when all attempts at compensation by the temperature regulatory center fail, the body loses heat like an inanimate object. Most patients with hypothermia exhibit tachycardia, tachypnea, hypotension, leukocytosis, acidosis, increased pulmonary wedge pressure, and right atrial pressure; patients with hypothermia caused by infection with bacteremia have much lower systemic vascular resistance and higher cardiac index than nonbacteremic patients with hypothermia.

Although most increases in body temperature seen in the clinic are fevers, changing lifestyles, old age, and medical treatments are responsible for an increasing number of patients with hyperthermia. Joggers and road runners are particularly prone to hyperthermia, especially during the summer months in areas where temperature is above 32.2°C (90°F) and humidity above 90%. Those at most risk fail to wear proper clothing, wear impermeable sweatsuits, or exercise after taking phenothiazines, anticholinergic drugs, or alcohol. Older people and those who previously have suffered from hyperthermia are particularly prone to hyperthermia because of the inability to respond normally to a heat load. Vacationers unaccustomed to saunas and hot tubs are another high-risk group for hyperthermia. Patients might not pay close enough attention to early signs of hyperthermia, such as headache, piloerection, and chills; and, at times, the first manifestations recognized (by others) are changes in gait, speech, or mental status. Transient paralysis or convulsions may be the first symptoms or signs.

There is no direct evidence in humans of a beneficial effect of fever in infectious states, but evolutionary arguments indicate that fever is important in enhancing inflammation. Additional evidence is available for the salutary effect of fever in cancer therapy. In contrast, the stress of a 13% increase in metabolic rate per degree of temperature Celsius (7% per degree of temperature Fahrenheit) is imposed on febrile patients. This stress may be detrimental to the elderly with cardiovascular disease, or to those with restrictive lung disease. Prolonged fever for over a week is often accompanied by a negative nitrogen balance and dehydration. Herpes simplex exacerbations (fever blisters), febrile convulsions, delirium, and albuminuria are also common.

Classically described fever patterns can be helpful in indicating the possible causes of infections. The graphic temperature chart can be indispensable to the clinician approaching the febrile patient. *Remittant* is the term used to describe a fever that fluctuates more than 1.1°C (2°F) daily but never returns to normal. Most fevers caused by infection are of this type. *Intermittent,* or quotidian, fever is characterized by wide swings in temperature each day with the peak usually in the afternoon and nadir in early morning. The temperature may be normal during the early and mid portion of the day. Intermittent fevers are often accompanied by rigors and profuse night sweats. Intermittent fevers are seen in patients with malaria, abscesses, and cholangitis. When characterized by very wide swings in temperature, intermittent fevers are termed *septic* or *hectic* fevers and indicate established deep abscess.

Sustained or continued fever, characterized by temperature elevation with little (less than 1°C) diurnal fluctuation, is seen in patients with pneumococcal pneumonia and typhoid fever. *Relapsing* indicates that bouts of fever are interspersed with afebrile periods of days to weeks. Examples of relapsing fever can be seen in Hodgkin's disease (Pel-Epstein fever), *P. vivax* malaria, *Borrelia* infection, and rat-bite fever due to *Spirillum minus.* In modern medicine, febrile pattern recognition is often unreliable because of purposeful or inadvertent use of analgesics, steroids, anti-inflammatory agents, and cooling devices that alter physiologic fever responses. In addition, uremics and quadraplegics may have blunted febrile responses, whereas patients with extensive surface burns or dermatologic conditions may have exaggerated and sustained fevers.

Inspection of the temperature chart should always include careful attention to the pulse and respiratory rate pattern. With most infections, the pulse rate will increase about 10 beats per minute for each 0.5 degree Celsius (each degree Fahrenheit) of temperature increase. The respiratory rate will also be above the normal 12 to 14 per minute, usually at 18 to 20 breaths per minute at rest. Three infectious diseases in which a relative bradycardia occurs are mycoplasma pneumonia, psittacosis, and typhoid fever.

Two special circumstances are often confusing to the clinician investigating a patient with fever. They are those patients with "factitious" fever and drug fever. Factitious, or feigned, fever is produced by the patient who, for reasons of secondary gain, is trying to simulate an organic illness. Two types of patients with factitious fever may be distinguished. One is the patient who is inducing fever by a self-inflicted disease such as a bacteremia or endotoxemia by injecting contaminated foreign debris. The other is the patient who has a spuriously high temperature due to manipulation of the thermometer. Clues for a "factitious" fever due to thermometer manipulation include a temperature of more than 41.1°C (106°F) in a patient who looks well, has no chills or rigors, shows no diurnal variations of temperature, has no tachycardia or tachypnea, and has no increase in the temperature of a freshly voided urine specimen. Patients with self-induced fever due to administration of a pyrogenic substance are often in the health care profession, appear well, have no weight loss, and have a normal physical examination between febrile episodes. Similarly, patients with drug (especially antibiotic) fever are confusing, especially when the antibiotic was appropriate and effective in eliminating the infection. These patients usually have sustained (sometimes intermittent) fever, appear entirely well, have an excellent appetite, and are inquiring about discharge from the hospital. Occasional findings supporting the diagnosis of drug fever include pruritus, the feeling of tingling or burning skin, and eosinophilia.

Most infectious causes of fever are naturally self-limited (such as viral diseases) or easily diagnosed and treatable bacterial diseases of the pharynx, ears, sinuses, upper respiratory airways, skin, and urinary tract. These usually are circumscribed illnesses of less than 7 to 10 days that do not require hospitalization and result in no long-term sequelae. Rarely is marked fever present for more than 3 to 4 days in these patients.

Fever persisting for more than 2 weeks, especially when it is accompanied by malaise, anorexia, and weight loss, requires thorough consideration and investigation. Often this will take place within a hospital setting with relatively invasive and expensive tests. Prerequisite to the development of an effective diagnostic plan is a detailed history and careful and complete physical examination with frequent, often daily, reassessment of selected parameters. In addition, a methodical and sequential laboratory evaluation is essential.

The special category of fever of unknown origin (FUO) refers to febrile diseases that have eluded diagnosis for 2 or 3 weeks despite a reasonably complete evaluation by physical examination, chest x-rays, routine blood tests, and cultures. The cause of prolonged fever in these patients will be determined about 90% of the time. Most will be common diseases that have manifested in an unusual way. Infections account for about one-third of FUOs. Miliary tuberculosis, bacterial endocarditis, biliary tract disease including liver abscess and viral hepatitis, pyelonephritis, abdominal abscess, osteomyelitis, and brucellosis are the major infections encountered. In adults, neoplasms are the cause of FUO in about 20% of cases. The fever is due to the neoplasm itself, and not to complicating infection. Lymphomas (Hodgkin's and non-Hodgkin's) and neoplasms (hepatoma, atrial myxoma, and hypernephroma) are frequent causes. Among the Hodgkin's lymphoma cases, those of the lymphocyte-depletion type in elderly patients are most likely to present as FUO. Collagen vascular diseases such as systemic lupus erythematosus, "juvenile" rheumatoid arthritis (Still's disease), polyarteritis, temporal arteritis, and polymyalgia rheumatica account for about 15% of FUO cases. Other causes of prolonged fever include granulomatous diseases such as sarcoidosis, idiopathic hepatitis, and inflammatory bowel disease.

An interesting group of patients who have perplexed clinicians are young individuals, especially women, with slightly exaggerated circadian rhythm, producing long-term daily afternoon temperatures of about 0.5°C higher than the normal. These women usually have no other associated manifestations of disease and are best served by long-term observation by one physician, who can provide reasonable periodic evaluation and reassurance to the patient and family.

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